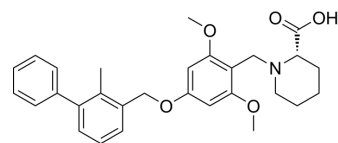


BMS-1

Cat. No.:	HY-19991
CAS No.:	1675201-83-8
Molecular Formula:	C ₂₉ H ₃₃ NO ₅
Molecular Weight:	475.58
Target:	PD-1/PD-L1; Apoptosis
Pathway:	Immunology/Inflammation; Apoptosis
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 2 years -20°C 1 year



SOLVENT & SOLUBILITY

In Vitro

Methanol : 25 mg/mL (52.57 mM; Need ultrasonic)
 DMSO : 7.14 mg/mL (15.01 mM; Need ultrasonic)
 DMF : 5 mg/mL (10.51 mM; Need ultrasonic)

	Solvent Concentration	Mass	1 mg	5 mg	10 mg
Preparing Stock Solutions	1 mM		2.1027 mL	10.5135 mL	21.0270 mL
	5 mM		0.4205 mL	2.1027 mL	4.2054 mL
	10 mM		0.2103 mL	1.0513 mL	2.1027 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

- Add each solvent one by one: 50% PEG300 >> 50% saline
Solubility: 10 mg/mL (21.03 mM); Clear solution; Need ultrasonic
- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
Solubility: ≥ 1 mg/mL (2.10 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
Solubility: ≥ 1 mg/mL (2.10 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil
Solubility: ≥ 1 mg/mL (2.10 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

BMS-1 is an inhibitor of the PD-1/PD-L1 protein/protein interaction (IC₅₀ between 6 and 100 nM)^{[1][2]}.

IC₅₀ & Target

PD1-PDL1^[1]

In Vitro

Since PD-1 mediated the exhaustion of natural killer (NK) cell by binding to its ligand PD-L1, BMS-1 (PD-1/PD-L1 inhibitor 1) (1 μ M, 3 days) is used to disturb the interaction between PD-1 and PD-L1. Dexamethasone induced increase of PD-1 expression and decrease of cytotoxicity of the co-cultured NK92 cells are reversed by BMS-1^[1]. BMS-1, a small-molecule immune checkpoint inhibitor of PD-1/PD-L1, can be used as a therapeutic strategy for tumor immunotherapy^[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Cytotoxicity Assay^[1]

Cell Line:	NK cells and HepG2 cells
Concentration:	1 μ M
Incubation Time:	3 days
Result:	Disturbed the interaction between PD-1 and PD-L1.

In Vivo

BMS-1 (500 μ g/mL; 100 μ L; i.p.) significantly increases the survival rates of the mVEGF165b group and mVEGF165b + MUC1 group^[3].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	BALB/c mice with EMT6 cells ^[3]
Dosage:	500 μ g/mL; 100 μ L
Administration:	i.p.
Result:	Increased the survival rates of the mVEGF165b group and mVEGF165b + MUC1 group. mVEGF165b combined with MUC1 results significant retardation of the tumor growth.

CUSTOMER VALIDATION

- Adv Funct Mater. 2021 Mar 21.
- Adv Sci (Weinh). 2024 Mar 9:e2310163.
- Acta Pharm Sin B. 2023 Aug 19.
- Small. 2022 Nov 11:e2205694.
- J Exp Clin Cancer Res. 2022 Jan 3;41(1):1.

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REFERENCES

- [1]. Zhao Y, et al. Depression Promotes Hepatocellular Carcinoma Progression through a Glucocorticoids Mediated Up-Regulation of PD-1 Expression in Tumor infiltrating NK Cells. Carcinogenesis. 2019 Feb 4.
- [2]. Li K, et al. Development of small-molecule immune checkpoint inhibitors of PD-1/PD-L1 as a new therapeutic strategy for tumour immunotherapy. J Drug Target. 2019 Mar;27(3):244-256.
- [3]. Zhang H, et al. Utilizing VEGF165b mutant as an effective immunization adjunct to augment antitumor immune response. Vaccine. 2019 Apr 3;37(15):2090-2098.
- [4]. Mengyuan Li, et al. KALRN mutations promote antitumor immunity and immunotherapy response in cancer. J Immunother Cancer. 2020 Oct;8(2):e000293.

Caution: Product has not been fully validated for medical applications. For research use only.

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